Alerts, Notices, and Case Reports

Bradytachyarrhythmia as the First Manifestation of Acute Rheumatic Carditis in an Adult

KAM S. WOO, MD
Shatin, Hong Kong

THE INCIDENCE OF rheumatic fever, a common and widespread disease in the late 19th and early 20th centuries, has been declining rapidly and seemingly disappeared in many Western countries, including the United Kingdom and North America, over the last 20 years.[1-3] A striking resurgence of acute rheumatic fever has been seen, however, in the intermountain areas of the continental United States and in Hawaii in recent years.[4-7] This case report alerts the medical community to the possibility of a widespread return of rheumatic fever, which many young physicians may never have encountered.[8-10]

Changing clinical manifestations of rheumatic fever, particularly in adults, have been reported in the United States and in Asian-Pacific countries over the last two decades, as compared with the classic pattern described in the literature.[11-14] Whereas various forms of bradytachyarrhythmias are not uncommonly encountered in clinical practice, this condition has seldom been documented as a dominant manifestation of acute rheumatic carditis. I report a patient presenting with such manifestations.

Report of a Case

A 55-year-old Chinese man was admitted to the Prince of Wales Hospital in April 1986 because of syncope with clouding of consciousness preceded by a 2-week history of general malaise and pyrexia but no sore throat. He had no history of rheumatic fever. On admission, he had a low-grade fever but no cardiac murmur. An electrocardiogram (ECG) revealed sinus tachycardia and complete atrioventricular (AV) block, with idiojunctional bradycardia at a rate of 35 per minute and a long period (3.6 seconds) of ventricular asystole (Figure 1). He had no response to intravenous atropine and, accordingly, temporary cardiac pacing was done. He had intermittent and self-remitting episodes of atrial fibrillation and flutter lasting for a few hours over the next three days (Figure 1). On day 3, he developed migratory polyarthritis involving the wrists, elbows, ankles, and shoulder joints. Two days later, pericarditis appeared, along with precordial pain, pericardial rub, and concave ST-segment elevation on the ECG (Figure 2).

The patient's laboratory results included a leukocyte count of 12,000 × 10^6 per liter (12,000 per cu mm), an erythrocyte sedimentation rate of 115 mm per h, and an antistreptolysin '0' titer of 333 U per ml (normal 240 U per ml). The results of serologic screening for rheumatoid factor, antinuclear factor, and elevated antivirus titers were negative. A throat swab culture found no evidence of streptococci. An echocardiogram confirmed the presence of pericardial effusion but had unremarkable intracardiac findings.

The patient's arthritis and pericarditis responded rapidly to rest and acetylsalicylate treatment (Table 1), which was started at 5.2 gm daily in divided doses and was tailed down over a period of 10 weeks. There was no clinical, radiologic, or echocardiographic evidence of cardiac decompensation. He remained in unstable supraventricular rhythm over the first 4 weeks, but stable sinus rhythm was restored by day 60. His erythrocyte sedimentation rate was normal at 6 months' follow-up. He has been followed regularly for 5 years and takes oral sulphonamide prophylactically because he is allergic to penicillin. He has had no relapse of cardiac arrhythmia or syncope, and there is no evidence of a residual valvular lesion on clinical examination or echocardiography.

Discussion

This patient had definite acute rheumatic fever diagnosed according to the revised Jones' criteria,[15] with polyarthritis, pericarditis, pyrexia, leukocytosis, a high erythrocyte sedimentation rate, and an elevated antistreptolysin '0' titer but no cardiac murmur (Table 2). Results of screening were negative for collagen vascular disease and viral infection. Tachyarrhythmias and conduction disorders, which closely followed the clinical course, appeared to be a consequence of the acute carditis. His subsequent progress with no recurrence of atrial fibrillation, flutter, or heart block on long-term follow-up supports this premise. This combination of arrhythmias in acute rheumatic fever has not been described previously.

Delayed AV conduction in the form of prolongation of the PR interval is a common feature of acute rheumatic fever. It is a minor diagnostic criterion and occurs with similar frequency in the polyarthritis of acute rheumatic fever with or without evidence of carditis.[16] High-grade AV block—especially of second degree (Wenckebach)—and, rarely, complete AV block in acute rheumatic carditis are well recognized. Their occurrence is less common, however, and usually of brief duration with no hemodynamic significance.[16]

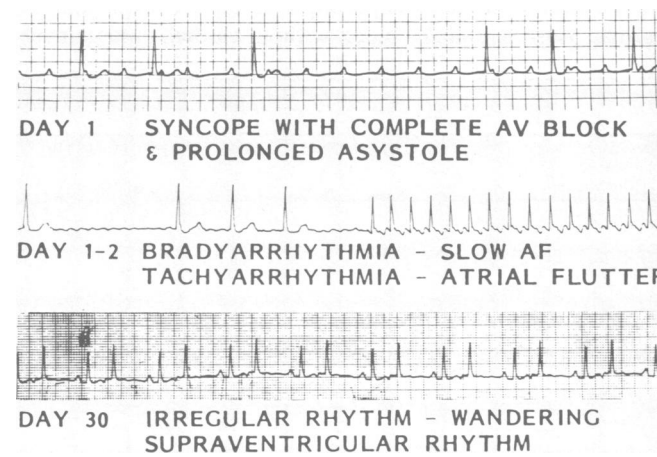

Figure 1.—Electrocardiographic rhythm strips show the bradytachyarrhythmias (day 1) and unstable supraventricular rhythm (day 30). AF = atrial fibrillation

(Woo KS: Bradytachyarrhythmia as the first manifestation of acute rheumatic carditis in an adult. West J Med 1992 Apr; 156:413-415)

From the Department of Medicine, the Chinese University of Hong Kong, Shatin, Hong Kong.

Reprint requests to Kam S. Woo, MD, Department of Medicine, Prince of Wales Hospital, Shatin, NT, Hong Kong.

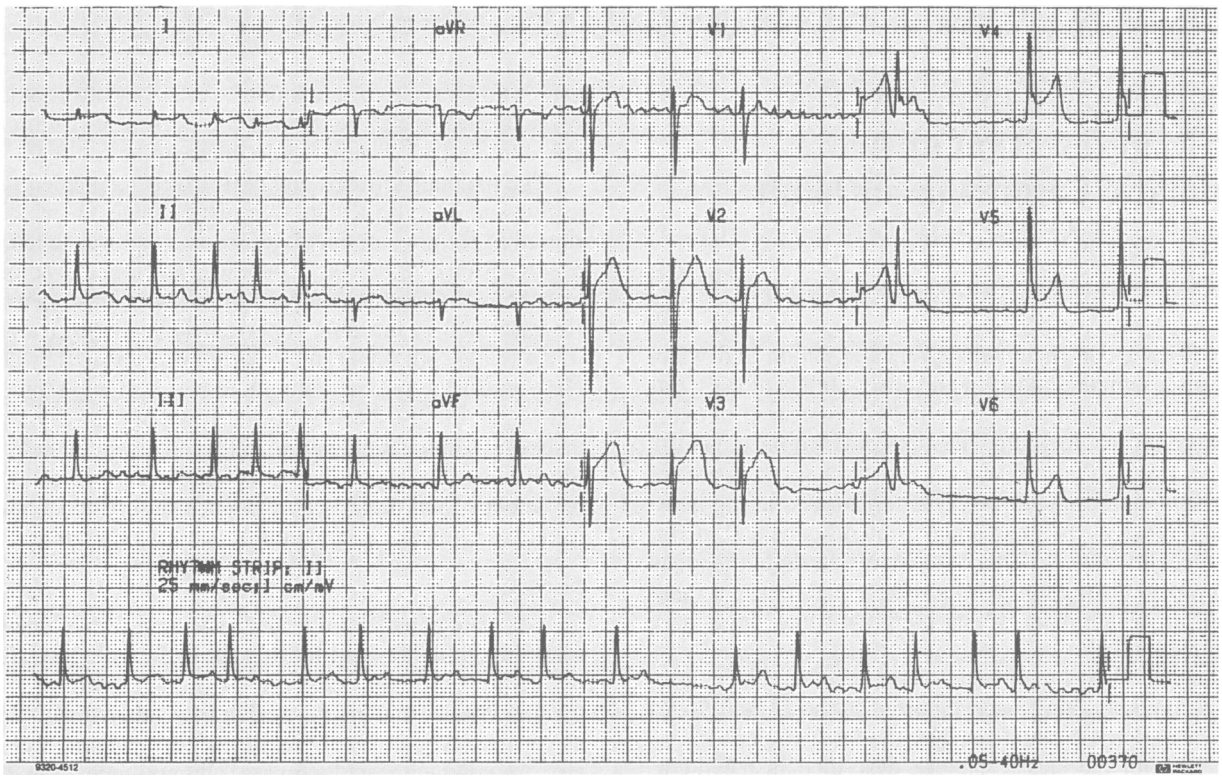

Figure 2.—Electrocardiogram on day 2 shows atrial fibrillation and concave ST-segment elevation in leads I, II, and V_2 to V_6, changes highly suggestive of pericardial inflammation.

The evanescence of heart block and its easy reversibility in most cases by administering atropine have previously been taken as evidence of a functional defect, rather than a structural lesion.[17] The bradyarrhythmias (complete AV block and prolonged ventricular asystole) in this patient, however, for which he was admitted to hospital and required temporary cardiac pacing, were more serious than the classic picture. This arrhythmic complication has not been reported previously and is highly suggestive of an organic involvement of conduction tissue by the acute rheumatic process.

Atrial fibrillation is thought to be a rare complication of acute rheumatic carditis, in contrast to its frequency in patients with longstanding mitral valvular disease with atrial enlargement and chronic atrial subendocardial inflammation.[18] This patient had episodes of tachyarrhythmia (atrial flutter and fibrillation) superimposed on a background of cardiac asystole and AV block without cardiac enlargement or cardiac decompensation. This suggests a direct atrial involvement. Aschoff bodies have been isolated in 21% to 55% of surgically excised left atrial appendages in patients with mitral stenosis.[19-20] The atrial tachyarrhythmias in this patient may relate to such pathologic abnormalities occurring in acute rheumatic carditis.

Acute rheumatic fever in the 1990s may differ from the classic patterns of the early 20th century.[11-14] More recent experience in Asian-Pacific regions and North America has revealed a lower incidence of carditis, erythema marginatum, chorea, and subcutaneous nodules but a higher incidence of polyarthritis and a higher proportion of adult patients. Acute rheumatic fever in adults recently has been characterized by dominant joint involvement while carditis, if present, is relatively mild.[11,12,14] My patient is unusual in that he presented with Stokes-Adams attacks as a result of his bradyarrhythmias. The underlying diagnosis of acute rheu-

TABLE 1.—*Progress of Patient With Acute Rheumatic Fever*

Date of Observation	*Fever*	*Arthritis*	*Cardiac Rhythm*	*Carditis*	*Erythrocyte Sedimentation Rate, mm/h*	*Antistreptolysin "O" titer, U/ml*
Day 1*	++	0	Complete heart block, supraventricular tachycardia	0	115	–
Day 3†	+	++	Atrial fibrillation	0	–	–
Day 5	+	+	Atrial fibrillation	Pericarditis	115	333
Day 10	0	0	Atrial fibrillation	Pericarditis	–	–
Day 20	0	0	Sinus	0	108	–
Day 30	0	0	Atrial fibrillation (intermittent)	0	55	–
Month 4	0	0	Atrial fibrillation (intermittent)	0	70	85
Month 6	0	0	Sinus	0	20-38	–

+ = mild intensity, ++ = moderate intensity, 0 = absent, – = not done

*Temporary cardiac pacing was performed on day 1.
†Treatment with rest and salicylate therapy was begun on day 3.

TABLE 2.—*Clinical Diagnosis of Acute Rheumatic Fever According to the Revised Jones' Criteria*

Revised Jones' Criteria	*This Patient*
Major	
Carditis	Pericarditis
Arthritis	Migratory polyarthritis
Erythema marginatum	Absent
Rheumatic nodule	Absent
Rheumatic chorea	Absent
Minor	
Fever	Low-grade fever
Prolonged PR interval in electrocardiogram	High-grade AV block
Leukocytosis	Present
High erythrocyte sedimentation rate	Erythrocyte sedimentation rate 110 mm/h
Previous rheumatic fever	Absent
Prerequisite	
Evidence of streptococcal infection	Antistreptolysin "O" titer 330 U/ml
Rheumatoid arthritis ruled out	Yes

AV = atrioventricular

matic fever with dominating carditis was confirmed by his subsequent development of polyarthritis and pericarditis. Such a combination of atrial fibrillation and AV block as a presentation of acute rheumatic fever has not been reported previously.

Bradytachyarrhythmias are encountered frequently in clinical practice, in particular among the geriatric population. Now that the medical community is facing a possible widespread return of rheumatic fever, this report highlights an atypical manifestation of the disorder and should alert clinicians to consider bradytachyarrhythmias as a presenting dominant manifestation of acute rheumatic carditis.

REFERENCES

1. Strasser T: Rheumatic fever and rheumatic heart disease in the 1970s. WHO Chron 1978; 32:18-25
2. Stollerman GH: Rheumatic Fever and Streptococcal Infection. New York, NY, Grune & Stratton, 1975, pp 63-99
3. DiSciascio G, Taranta A: Rheumatic fever in children. Am Heart J 1980; 99:635-658
4. Veasy LG, Wiedmeier SE, Orsmond GS, et al: Resurgence of acute rheumatic fever in the intermountain area of the United States. N Engl J Med 1987; 306:421-427
5. Congeni B, Rizzo C, Congeni J, Sreenivassan VV: Outbreak of acute rheumatic fever in northeast Ohio. J Pediatr 1987; 111:176-179
6. Hosier DM, Craenen JM, Teske DW, Wheller JJ: Resurgence of acute rheumatic fever. Am J Dis Child 1987; 141:730-733
7. Chun LT, Reddy DV, Yamamoto LG: Rheumatic fever in children and adolescents in Hawaii. Pediatrics 1987; 79:549-552
8. Giardina AC: Resurgence of acute rheumatic fever. N Engl J Med 1987; 317:507-508
9. Bissenden JG: Transatlantic warning bells sound on rheumatic fever. Br Med J 1989; 296:1215
10. Bland EF: Rheumatic fever: The way it was. Circulation 1987; 76:1190-1213
11. Barnet AL, Terry EE, Persellin RH: Acute rheumatic fever in adults. JAMA 1975; 232:925-928
12. Persellin RH: Acute rheumatic fever: Changing manifestations [Editorial]. Ann Intern Med 1978; 89:1002-1003
13. Majeed HA, Khan N, Dabbagh M, Najdi K, Khateeb N: Acute rheumatic fever during childhood in Kuwait: The mild nature of the initial attack. Ann Tropic Pediatr 1981; 1:13-20
14. Woo KS, Kong SM, Wai KH: The changing prevalence and pattern of acute rheumatic fever and rheumatic heart disease in Hong Kong (1968-1978). Aust NZ J Med 1983; 13:151-156
15. Ad Hoc Committee to Revise the Jones' Criteria (modified), Council on Rheumatic Fever and Congenital Heart Disease of the American Heart Association: Jones' criteria (revised) for guidance in the diagnosis of rheumatic fever. Circulation 1984; 69:204A-208A
16. Stollerman GH: Clinical manifestations of acute rheumatic fever. *In* Rheumatic Fever and Streptococcal Infection. New York, NY, Grune & Stratton, 1975, pp 147-177
17. Robinson RW: Effect of atropine upon the prolongation of the P-R interval found in acute rheumatic fever and certain vagotonic persons. Am Heart J 1945; 29:378-383
18. Stollerman GH: Connective tissue diseases of the cardiovascular system. *In* Braunwald E (Ed): Heart Disease—A Textbook of Cardiovascular Medicine. Philadelphia, Pa, Saunders, 1980, pp 1734-1735
19. Thomas WA, Averill JH, Castleman B, Bland EF: The significance of Aschoff bodies in the left atrial appendage. N Engl J Med 1953; 249:761
20. Virmani R, Roberts WC: Aschoff bodies in operatively excised atrial appendages and in papillary muscles—Frequency and clinical significance. Circulation 1977; 55:559-563

Tularemia Presenting as an Isolated Pleural Effusion

LINDA M. FUNK, MD
STEVEN Q. SIMPSON, MD
GREGORY MERTZ, MD
Albuquerque, New Mexico

JOHN BOYD, MD
Durango, Colorado

ALTHOUGH TULAREMIA has declined in incidence since it was first described by Edward Francis in 1925,[1] it continues to occur both sporadically and in epidemics in the United States.[2] There are several forms of tularemia, but it is the pneumonic form that is often mistaken for other, more common diseases. Because pulmonary tularemia is the most severe form, with the highest reported rates of mortality,[3] its recognition and treatment by physicians in and around endemic areas are important. We report a case of tularemia presenting as pleurisy in a young woman.

Report of a Case

The patient, a 37-year-old woman, was referred to the University of New Mexico Medical Center (Albuquerque) from her primary physician in southwestern Colorado, where she had been seen in mid-July 1991 with fever, chills, and pleuritic chest pain of two days' duration. Other presenting symptoms included diffuse myalgias, headache, night sweats, and a nonproductive cough. The results of chest

(Funk LM, Simpson SQ, Mertz G, Boyd J: Tularemia presenting as an isolated pleural effusion. West J Med 1992 Apr; 156:415-417)

From the Pulmonary Division (Drs Funk and Simpson), and the Division of Infectious Disease (Dr Mertz), Department of Medicine, University of New Mexico, Albuquerque. Dr Boyd is in private practice in Durango, Colorado.

Reprint requests to Linda Funk, MD, Pulmonary Division, Department of Medicine, 5th Flr, Ambulatory Care Center, University of New Mexico, 2211 Lomas Blvd NE, Albuquerque, NM 87131-5271.